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Côme Roux, Béatrice Villemur, Brigitte Giovannoni, Lucie Koeyemelk, Monique Mendelson, Meriem Benmerad, Marie Joyeux-Faure, Renaud Tamisier, Jean-Louis Pepin

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1 **Prevalence of obstructive sleep apnea syndrome in patients with lymphedema referred for**  
2 **complete decongestive therapy**

3

4 **Short title: Obstructive sleep apnea in lymphedema patients**

5

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5 supported by the French National Research Agency (Agence Nationale de la Recherche) in the  
6 framework of the "Investissements d’avenir” program (ANR-15-IDEX-02).

1 **ARTICLE HIGHLIGHTS**

2 **Type of Research:** Single center prospective study

3 **Key Findings:** Obstructive sleep apnea was prevalent in 74% of 43 patients with lymphedema presenting  
4 for complete decongestive therapy.,particularly in those with lower limb lymphedema (85% prevalence).

5 **Take home Message:** Overweight and obese patients with lymphedema should be systematically  
6 screened for obstructive sleep apnea.

7

8 **Table of Contents Summary**

9 Obstructive sleep apnea was prevalent in 74% of 43 patients with lymphedema presenting for complete  
10 decongestive therapy. The authors suggest systematic screening of obstructive sleep apnea in overweight-  
11 obese patients with lymphedema.

12

## 1 ABSTRACT

2 **Objectives:** Obstructive sleep apnea (OSA) syndrome is one of the most frequent chronic  
3 diseases in the general population. The nocturnal rostral fluid shift is accepted as a key  
4 mechanism in OSA pathogenesis in medical conditions associated with fluid overload. The main  
5 objective of this study was to assess the prevalence of OSA in patients with lymphedema.

6 **Material and methods:** A prospective study was conducted in patients with lymphedema  
7 hospitalized for complete decongestive therapy between December 2016 and November 2017. A  
8 nocturnal respiratory polygraphy, an Epworth sleepiness scale and lymphedema volume  
9 measurements were performed on the first day of decongestive therapy. OSA was diagnosed in  
10 patients with an apnea-hypopnea index (AHI)  $\geq 5$  events/h and patients who had been previously  
11 diagnosed with OSA and treated by continuous positive airway pressure were identified.

12 **Results:** Forty-three patients completed the study. The prevalence of OSA was 74% and was  
13 higher for patients with lymphedema of the lower limbs (85%) versus upper limbs (56%). OSA  
14 severity was correlated with a marker of lower limb lymphedema severity ( $p = 0.012$ ). The body  
15 mass index (BMI) was higher than in OSA patients than non OSA patients (31.0 [25.3 ; 37.0]  
16 versus 24.2 [22.7 ; 26.7]  $\text{kg/m}^2$  (median, 25<sup>th</sup> ; 75th percentiles),  $p < 0.01$ ). Hypertension was  
17 prevalent in 47% of patients with OSA vs 18% in non OSA patients.

18 **Conclusion:** Patients with lymphedema presenting for complete decongestive therapy present a  
19 high prevalence rate of OSA, particularly in those with lower limb lymphedema. OSA severity  
20 was correlated with a marker of lower limb lymphedema severity. These results suggest that  
21 overweight and obese patients with lymphedema should be systematically screened for OSA.

22

23 ClinicalTrials.gov: n° NCT02979184.

1 **KEY WORDS**

2 Lymphedema; Obstructive Sleep Apnea; Complete decongestive therapy; Fluid shift

## 1 INTRODUCTION

2 Lymphedema is the result of intra-tissue fluid accumulation caused by inability of the lymphatic  
3 system to absorb excess fluid from the venulo-capillary circulation. Treatment of lymphedema  
4 with complete decongestive therapy (CDT), which includes manual lymphatic drainage,  
5 lymphedema rehabilitation exercises, compression therapy and skin care, has been shown to  
6 reduce lymphedema volume by 45-70%<sup>1-3</sup>.

7 Obstructive sleep apnea is a common sleep disorder which is characterized by repetitive episodes  
8 of upper airway obstruction with persistence of respiratory efforts. The prevalence of obstructive  
9 sleep apnea (OSA) defined by an apnea-hypopnea index (AHI)  $\geq 5$  events per hour is estimated  
10 between 9% and 38% of the general population<sup>4,5</sup>. The prevalence of OSA is increased in  
11 patients with conditions characterized by fluid-retaining states such as heart failure and end-stage  
12 renal disease<sup>6-9</sup>. Lower-limb fluid accumulation, and its subsequent overnight rostral  
13 redistribution, has emerged as a contributor to the pathophysiology of OSA<sup>10</sup>. The prevention of  
14 fluid accumulation during the day, using compression stockings or by regular exercise has also  
15 been shown to reduce the frequency and severity of OSA<sup>11-13</sup>.

16 To our knowledge, no study has examined the prevalence of OSA in patients with lymphedema  
17 or the effects of complete decongestive therapy on OSA in this group of patients.

18 Therefore the aim of the present study was to test the hypothesis that lymphedema could be a  
19 factor predisposing to OSA, through an increase in the fluid shift. The secondary objectives were  
20 to assess the correlation between a marker of the severity of lymphedema (i.e. limb volume) and  
21 the severity of OSA, as well as to analyze the impact of CDT on OSA.

## 1 MATERIAL AND METHODS

2 The study was conducted in accordance with applicable good clinical practice requirements in  
3 Europe, French Law, ICH E6 recommendations, and Ethical Principles of the Helsinki  
4 Declaration (South Africa 1996 and Edinburgh 2000). It was approved by an independent Ethics  
5 Committee (Comité de Protection des Personnes, Grenoble, France, IRB0005578) and registered  
6 on the ClinicalTrials.gov site (NCT02979184). Written informed consent was obtained from all  
7 patients. An external data quality control was performed systematically for the following criteria:  
8 informed consent, complications, adverse events and case report forms.

### 9 Study design

10 We conducted a prospective study evaluating the prevalence of OSA in patients with  
11 lymphedema hospitalized in the Vascular Rehabilitation Unit of the University Hospital  
12 Grenoble Alps (CHUGA). Lymphedema was diagnosed clinically by thorough evaluation and  
13 physical examination. Circumferential (>2 cm) differences between the affected and non-  
14 affected extremity were performed to confirm the diagnosis<sup>14</sup>.

15 Patients between 18 and 85 years with lymphedema of one or more limbs, hospitalized in the  
16 Vascular Rehabilitation unit of the CHUGA for complete decongestive therapy were included.  
17 Non-inclusion criteria included patients under legal protection and those presenting  
18 contraindications to CDT (acute limb infection, active venous thrombosis, uncontrolled heart  
19 failure and hypertension, acute renal failure, obstruction of the superior vena cava, untreated  
20 active malignant tumor, peripheral arterial obstructive disease with systolic pressure index < 0.5,  
21 advanced diabetic microangiopathy). We also excluded patients with a popliteal aneurysm (more  
22 than 1.6 cm) to avoid any rupture with pressotherapy.

1 The main objective of the study was to determine the prevalence of OSA, defined by an AHI  $\geq$  5  
2 events/h or the presence of previously diagnosed OSA. The secondary endpoints assessed were  
3 factors associated with the presence of OSA such as the correlation between markers of the  
4 severity of lymphedema and OSA, and the evolution of AHI, lymphedema volumes and BMI,  
5 before and after rehabilitation.

### 6 **Complete decongestive therapy**

7 Patients received complete decongestive therapy for a total of 12 days in an inpatient facility.  
8 CDT included daily sessions of manual lymphatic drainage, pressotherapy followed by the  
9 application of bandages and exercise.

### 10 **Lymphedema volume measurements**

11 The volume of the lower and upper limbs was calculated from the perimeter measurements taken  
12 every 5 cm according to the Frustum method. The lymphedema severity criterion used was the  
13 difference in volume between the two affected limbs. Only unilateral lymphedema was taken  
14 into account.

### 15 **Sleep study**

16 With the exception of those already treated by CPAP for known OSA, all patients had a sleep  
17 study (respiratory polygraphy) in the first night following admission for rehabilitation. All  
18 recordings were scored by a sleep specialist to calculate apnea-hypopnea index (AHI). Patients  
19 with an AHI  $\geq$  5 events/h underwent a follow-up polygraphy at the end of the rehabilitation  
20 period.

### 21 **Statistical analysis**

22 Statistical analyses were performed using SAS® v9.4 software. The 95% confidence interval of  
23 the prevalence of OSA was calculated using the Wilson formula with continuity correction.

1 Inter-group comparisons were made by multivariate analysis with an accurate Fischer test for  
2 qualitative variables and a non-parametric Mann Whitney test for quantitative variables. The  
3 correlations were studied using the Spearman correlation coefficient. The evolution of the  
4 parameters before and after rehabilitation was evaluated by the Wilcoxon signed-ranks test.

## 1 **RESULTS**

2 Forty-five patients were included between December 2016 and November 2017 (Figure 1, Table  
3 I). Two patients withdrew during the study. Among the remaining 43 patients, 32 patients had  
4 OSA (6 previously diagnosed OSA treated by CPAP and 26 newly diagnosed for OSA with an  
5 AHI  $\geq 5$  events/h).

### 6 **Population characteristics**

7 The prevalence of OSA was 74% (n=32/43) in the entire population. It was 85% (n=23/27) in  
8 patients with lower limb lymphedema and 56% (n=9/16) in patients with upper limb  
9 lymphedema.

10 For the 26 newly diagnosed OSA patients, the median AHI was 13.1 [7.7 ; 20.7] events/h  
11 (median, 25<sup>th</sup> ; 75<sup>th</sup> percentiles). Among them, 4 (15%) had severe OSA (AHI  $\geq 30$ /h) 9 (35%)  
12 had moderate OSA ( $15 \leq$  AHI  $< 30$  events/h) and 13 (50%) had mild OSA ( $5 \leq$  AHI  $< 15$   
13 events/h).

14 The median BMI of OSA patients was higher than in non-apneic patients (31.0 [25.3 ; 37.0]  
15 versus 24.2 [22.7 ; 26.7] kg/m<sup>2</sup>, (p<0.01)).

### 16 **Correlation between OSA and lymphedema**

17 There was a positive correlation between the severity of OSA (AHI) and a marker of the severity  
18 of lower limb lymphedema (the difference in volume between the two affected limbs)  
19 (correlation coefficient: 0.667; p=0.016) (Figure 2). This correlation did not exist between the  
20 severity of OSA and a marker of the severity of upper limb lymphedema (Correlation coefficient:  
21 0.045; p=0.878) (Figure 3).

### 22 **Effect of complete decongestive therapy**

1 There was no significant change in the AHI after CDT ( $p=0.46$ ). During CDT, the volume of the  
2 most affected lymphedematous limb decreased by a median of 10.3% in OSA patients  
3 ( $p<0.0001$ ) and the BMI by 0.38 kg/m<sup>2</sup> ( $p<0.0001$ ), (Figure 4 and table II).  
4 There was no correlation between changes in AHI and BMI (correlation coefficient: -0.05,  
5  $p=0.85$  or between changes in AHI and volumes (correlation coefficient: -0.27,  $p=0.40$ ),  
6 following CDT.  
7 In the subgroup of patients with OSA ( $n=32$ ), BMI and limb volume were significantly reduced  
8 after CDT, both  $p<0.01$  (Table III). In the subgroup of patients without OSA ( $n=11$ ), BMI and  
9 limb volume also decreased significantly ( $p=0.022$  and  $p<0.01$ , respectively).

## 1 DISCUSSION

2 In the present study, 74% of patients with lymphedema presented at least mild OSA (AHI>5  
3 events/hr). The prevalence reached 86% in patients with lower limb lymphedema (85%), despite  
4 a predominantly female population. This prevalence rate is by far higher than what is estimated  
5 in the general population (i.e. between 9 and 38% of the general population)<sup>15</sup>. However, such  
6 OSA prevalence is similar to what is observed in patients with fluid-retaining states such as heart  
7 failure and end-stage renal disease<sup>6-9</sup>.

8 The higher prevalence of OSA in lower limb lymphedema and the existence of a correlation  
9 between the severity of lower limb lymphedema (volume-related) and the severity of OSA  
10 support the role of fluid shift in promoting OSA and also the potential presence of fluid-retaining  
11 disorders in these patients. The nocturnal rostral redistribution of fluid is known to play an  
12 important role in the pathophysiology of OSA by increasing peri-pharyngeal edema, reducing  
13 upper airway (UA) size and increasing UA resistance and collapsibility. In the present study,  
14 beyond fluid shift, factors associated with the presence of OSA were obesity, age and prevalent  
15 hypertension, which is consistent with previous studies<sup>16-18</sup>. Future prospective studies in a  
16 larger sample sizes with a better documentation of the contribution of the nocturnal fluid shift to  
17 the pathophysiology of OSA in patients with lymphedema are required. A precise  
18 characterization of fluid overload by bio-electrical impedance in different body segments would  
19 be contributive<sup>19</sup>.

20 We did not observe a significant change in the AHI after the 12 day CDT period. However,  
21 follow-up sleep studies were done during the last night off CDT, following acute mobilization of  
22 fluid by complete decongestive therapy<sup>1</sup>. One can hypothesize that the effect of CDT on OSA

1 could be masked at this stage. It would be interesting to perform final evaluation during a more  
2 stable state, away from complete decongestive therapy.

3 OSA and its cardiovascular consequences have been widely explored in observational and  
4 prospective studies. Population-based and sleep-clinic-based cohort studies have shown an  
5 association between obstructive sleep apnea and hypertension, coronary artery disease, atrial  
6 fibrillation, stroke and heart failure <sup>20</sup>. There is a consensus in the OSA field for increasing  
7 screening in at risk populations with the main goal of improving symptoms, quality of life and  
8 reducing cardiovascular risk. The treatment of OSA in this population should also be encouraged  
9 in order to favor patients' engagement in rehabilitation procedures. The management of  
10 overweight and obesity can also contribute to reducing the risk of OSA <sup>21</sup>. The effects of the  
11 different modalities of decongestive therapy on the reduction of fluid shift should also be  
12 explored in future studies to determine which modality is the most efficient.

13 In the present study, we did not explore the association between OSA and thrombosis. However,  
14 the current epidemiological evidence supports the hypothesis that OSA may be a risk factor for  
15 venous thromboembolism <sup>22</sup>. Future studies in patients with lymphedema are needed to further  
16 explore this association.

17

### 18 **Limitations of the study**

19 There are several limitations to our study. We included patients with lymphedema admitted for  
20 complete decongestive therapy in a hospital setting. Therefore, our sample size is subject to  
21 selection bias and this limits the generalization of our findings. Future studies designed to  
22 include patients who are seen both in inpatient and outpatient facilities should be conducted to  
23 further explore the prevalence of OSA in patients with lymphedema.

1

**2 CONCLUSION**

3 The present study confirms that patients with lymphedema presenting for complete decongestive  
4 therapy present a high prevalence rate of OSA. We also observed a significant correlation  
5 between ~~the~~ a marker of the severity of lower limb lymphedema and the severity of OSA. Our  
6 results suggest a systematic screening of OSA in overweight and obese patients with  
7 lymphedema. Further studies are needed in a larger sample size to determine whether or not  
8 patients with a BMI<25 require systematic screening as well. Further prospective studies with  
9 larger sample sizes are needed to confirm the role of fluid shift in the pathophysiology of OSA in  
10 patients with lower limb lymphedema and define appropriate management programs.

## 1 REFERENCES

- 2 1 Pereira De Godoy JM, Franco Brigidio PA, Salles Cunha SX, Batigalia F, De Fatima  
3 Guerreiro Godoy M. Mobilization of fluids in large volumetric reductions during intensive  
4 treatment of leg lymphedema. *International angiology : a journal of the International Union of*  
5 *Angiology*. 2013; 32: 479-82.
- 6 2 Quere I, Presles E, Coupe M, Vignes S, Vaillant L, Eveno D, *et al*. Prospective  
7 multicentre observational study of lymphedema therapy: POLIT study. *Journal des maladies*  
8 *vasculaires*. 2014; 39: 256-63.
- 9 3 Tzani I, Tsihlaki M, Zerva E, Papatthasiou G, Dimakakos E. Physiotherapeutic  
10 rehabilitation of lymphedema: state-of-the-art. *Lymphology*. 2018; 51: 1-12.
- 11 4 Levy P, Kohler M, McNicholas WT, Barbe F, McEvoy RD, Somers VK, *et al*.  
12 Obstructive sleep apnoea syndrome. *Nature reviews Disease primers*. 2015; 1: 15015.
- 13 5 Peppard PE, Young T, Barnet JH, Palta M, Hagen EW, Hla KM. Increased prevalence of  
14 sleep-disordered breathing in adults. *Am J Epidemiol*. 2013; 177: 1006-14.
- 15 6 Logan AG, Perlikowski SM, Mente A, Tisler A, Tkacova R, Niroumand M, *et al*. High  
16 prevalence of unrecognized sleep apnoea in drug-resistant hypertension. *Journal of hypertension*.  
17 2001; 19: 2271-7.
- 18 7 Lyons OD, Bradley TD, Chan CT. Hypervolemia and Sleep Apnea in Kidney Disease.  
19 *Seminars in nephrology*. 2015; 35: 373-82.
- 20 8 Worsnop CJ, Naughton MT, Barter CE, Morgan TO, Anderson AI, Pierce RJ. The  
21 prevalence of obstructive sleep apnea in hypertensives. *American journal of respiratory and*  
22 *critical care medicine*. 1998; 157: 111-5.

- 1 9 Yumino D, Wang H, Floras JS, Newton GE, Mak S, Ruttanaumpawan P, *et al.*  
2 Prevalence and physiological predictors of sleep apnea in patients with heart failure and systolic  
3 dysfunction. *Journal of cardiac failure.* 2009; 15: 279-85.
- 4 10 White LH, Bradley TD, Logan AG. Pathogenesis of obstructive sleep apnoea in  
5 hypertensive patients: role of fluid retention and nocturnal rostral fluid shift. *Journal of human*  
6 *hypertension.* 2015; 29: 342-50.
- 7 11 Mendelson M, Lyons OD, Yadollahi A, Inami T, Oh P, Bradley TD. Effects of exercise  
8 training on sleep apnoea in patients with coronary artery disease: A randomised trial. *European*  
9 *Respiratory Journal.* 2016; 48: 142-50.
- 10 12 Perger E, Jutant EM, Redolfi S. Targeting volume overload and overnight rostral fluid  
11 shift: A new perspective to treat sleep apnea. *Sleep medicine reviews.* 2018; 42: 160-70.
- 12 13 White LH, Bradley TD. Role of nocturnal rostral fluid shift in the pathogenesis of  
13 obstructive and central sleep apnoea. *The Journal of physiology.* 2013; 591: 1179-93.
- 14 14 Kim M, Shin KH, Jung SY, Lee S, Kang HS, Lee ES, *et al.* Identification of Prognostic  
15 Risk Factors for Transient and Persistent Lymphedema after Multimodal Treatment for Breast  
16 Cancer. *Cancer research and treatment : official journal of Korean Cancer Association.* 2016; 48:  
17 1330-7.
- 18 15 Senaratna CV, Perret JL, Matheson MC, Lodge CJ, Lowe AJ, Cassim R, *et al.* Validity of  
19 the Berlin questionnaire in detecting obstructive sleep apnea: A systematic review and meta-  
20 analysis. *Sleep medicine reviews.* 2017; 36: 116-24.
- 21 16 Franklin KA, Lindberg E. Obstructive sleep apnea is a common disorder in the  
22 population-a review on the epidemiology of sleep apnea. *J Thorac Dis.* 2015; 7: 1311-22.

- 1 17 Hla KM, Young TB, Bidwell T, Palta M, Skatrud JB, Dempsey J. Sleep apnea and  
2 hypertension. A population-based study. *Annals of internal medicine*. 1994; 120: 382-8.
- 3 18 Young T, Peppard PE, Taheri S. Excess weight and sleep-disordered breathing. *Journal of*  
4 *applied physiology* (Bethesda, Md : 1985). 2005; 99: 1592-9.
- 5 19 Kyle UG, Bosaeus I, De Lorenzo AD, Deurenberg P, Elia M, Manuel Gomez J, *et al.*  
6 *Bioelectrical impedance analysis-part II: utilization in clinical practice. Clinical nutrition*  
7 *(Edinburgh, Scotland)*. 2004; 23: 1430-53.
- 8 20 Drager LF, McEvoy RD, Barbe F, Lorenzi-Filho G, Redline S, Initiative I. Sleep Apnea  
9 and Cardiovascular Disease: Lessons From Recent Trials and Need for Team Science.  
10 *Circulation*. 2017; 136: 1840-50.
- 11 21 Peppard PE, Young T, Palta M, Dempsey J, Skatrud J. Longitudinal study of moderate  
12 weight change and sleep-disordered breathing. *Jama*. 2000; 284: 3015-21.
- 13 22 Garcia-Ortega A, Manas E, Lopez-Reyes R, Selma MJ, Garcia-Sanchez A, Oscullo G, *et*  
14 *al.* Obstructive sleep apnoea and venous thromboembolism: pathophysiological links and clinical  
15 implications. *The European respiratory journal*. 2019; 53.

## **FIGURE LEGENDS**

Figure 1. Study flow chart

Figure 2. Severity of OSA (AHI) according to the lower limb lymphedema (Difference in volume between affected and healthy limbs). Correlation coefficient: 0.667;  $p=0.016$ .

Figure 3. Severity of OSA (AHI) according to the upper limb lymphedema (Difference in volume between affected and healthy limbs). Correlation coefficient: 0.045;  $p=0.878$ .

Figure 4. Patient with upper and lower limb lymphedema, before (A1, B1) and after (A2, B2) 12 days of complete decongestive therapy

## TABLES

Table 1. Population characteristics

	No OSA n=11	OSA n=32	p
Age (years)	55.9 [43.5 ; 70.1]	68.6 [59.9 ; 74.8]	0.12
Number of women (n (%))	8 (73)	23 (72)	1.00
BMI (kg/m <sup>2</sup> )	24.2 [22.7 ; 26.7]	31.0 [25.3 ; 37.0]	<0.01
Diabetes (n (%))	0 (0)	4 (13)	0.56
Tobacco use (n (%))	1 (9)	10 (31)	0.24
Hypertension (n (%))	2 (18)	15 (47)	0.15
ESS score	7 [3 ; 9]	9 [2 ; 11]	0.46
Lymphedema of lower limb	4 (36)	23 (72)	0.07
Lymphedema of upper limb	7 (64)	9 (28)	
Maximum volume of lower limb (ml)*	9003 [8556; 9985]	9128 [7221 ; 12227]	0.97
Maximum volume of upper limb (ml)*	3193 [2400; 3731]	2864 [2713 ; 3615]	0.84

BMI, body mass index; ESS, Epworth sleepiness scale

\* Volume of the lower or upper limb on the side most affected by lymphedema.

Data are expressed as median [25th; 75th percentiles] or n (%).

Table 2. Evolution of AHI, BMI and volumes after rehabilitation in OSA patients

	Number of patients	Difference after-before rehabilitation	p
Maximum volumes (%)*	31	-10.3 [-14.8 ; -6.0]	<0.0001
BMI (kg/m <sup>2</sup> )	32	-0.38 [-1.45 ; -0.27]	<0.0001
AHI (events/h)	13	0.9 [-1.5 ; 7.4]	0.46
ESS score	20	0.0 [-1.0 ; 0.5]	0.18

AHI, apnea-hypopnea index; BMI, body mass index; ESS, Epworth sleepiness scale.

Data are expressed as median [25th ; 75th percentiles]

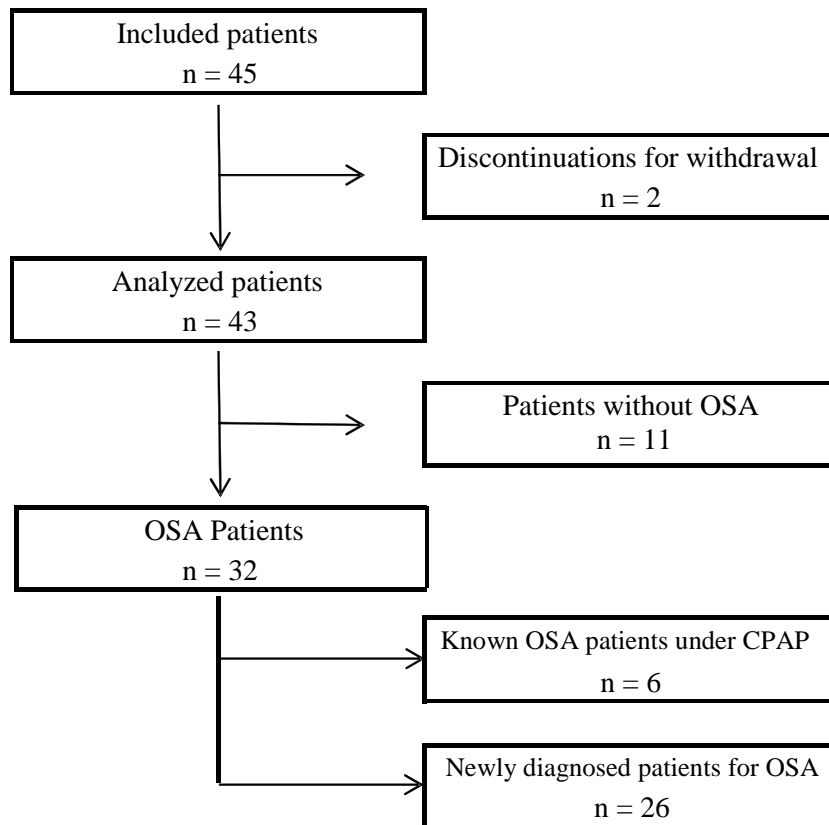
\*Maximum volume corresponded to the variation (%) of the most affected limb for uni- or bilateral lymphedema.

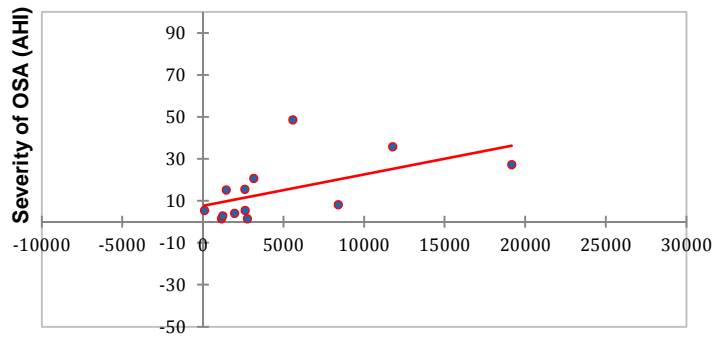
Table 3. Evolution of BMI and limb volume following rehabilitation in OSA and non OSA groups

	OSA (n=32)			No OSA (n=11)		
	Before	After	p	Before	After	P
BMI (kg/m <sup>2</sup> )	31.0 [25.4; 36.3]	29.9 [25.7; 35.6]	<0.000 1	24.2 [23.2; 26.5]	24.1 [22.4; 26.5]	0.022
Limb volume (mL)	7293 [3783; 10289]	6577 [3049; 9031]	<0.000 1	3731 [3007; 8556]	3029 [2647; 8164]	0.001

BMI, body mass index.

Data are expressed as median [25th ; 75th percentiles]





(Vmax-Vmin) of lower limb lymphedema (ml)

